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Original Article

Paediatrics Section

Clinical Profile and Outcome of Paediatric Patients with Diabetic Ketoacidosis: A Retrospective Cohort Study

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ABSTRACT

Introduction: Diabetic Ketoacidosis (DKA) remains a life-threatening metabolic complication in children with Type 1 Diabetes Mellitus (T1DM), particularly in resource-limited settings. Early diagnosis and prompt management are vital for improving outcomes.

Aim: To assess the clinical profile, laboratory parameters and outcomes of paediatric patients presenting with DKA and to identify factors associated with mortality.

Materials and Methods: This retrospective cohort study included 40 paediatric patients diagnosed with DKA and admitted to the Paediatric Intensive Care Unit (PICU) of Malda Medical College and Hospital, Malda, West Bengal, India. Children under 12 years who were admitted with a diagnosis of DKA between January 1, 2022 and December 31, 2024, were included. Clinical history, physical examination findings, laboratory values and treatment outcomes were recorded. Data were analysed using Statistical Package for the Social Sciences (SPSS) software and associations between various parameters and outcomes were evaluated using appropriate statistical tests.

Results: The highest proportion of DKA cases, 17 (42.5%), occurred in children aged 9 to 12 years. Both sexes were equally affected. Most patients, 21 (52.5%), belonged to a low Socio-economic Status (SES). Common symptoms included vomiting in 29 (72.5%), polyuria in 27 (67.5%) and polydipsia in 23 (57.5%). Dehydration was observed in 34 (85%) and Kussmaul breathing in 26 (65%) of the patients. The mean arterial pH was 7.12±0.14 and the mean HbA1c was 5.61±0.70%. The mortality rate was 10% (n=4). Significant predictors of poor outcomes included low SES (p-value=0.004), a positive family history of diabetes (p-value=0.049), lower pH (p-value=0.0016), higher HbA1c (p-value=0.0035), elevated serum urea (p-value=0.0049) and creatinine (p-value=0.0021).

Conclusion: Paediatric DKA continues to pose a serious clinical challenge. SES, family history and the degree of metabolic derangement significantly impact outcomes. Early recognition and timely management are critical in reducing mortality and improving prognosis.

Keywords: Hyperglycaemia, Metabolic acidosis, Mortality predictors, Type 1 diabetes mellitus

INTRODUCTION

The DKA is one of the most serious acute complications of T1DM in children and is associated with significant morbidity and mortality if not managed promptly and appropriately. DKA results from an absolute or relative deficiency of insulin, combined with an increase in counter-regulatory hormones such as glucagon, catecholamines, cortisol and growth hormone. This leads to hyperglycaemia, ketosis and metabolic acidosis [1]. The incidence of DKA as the first presentation of type 1 diabetes is especially high in low- and middle-income countries due to delayed diagnosis, lack of awareness and inadequate access to healthcare [2]. Globally, the frequency of DKA at the time of diagnosis of diabetes varies significantly, ranging from 15-70%, depending on regional healthcare infrastructure and awareness [3]. In India, several studies have reported the incidence of DKA in newly diagnosed paediatric diabetes cases to be between 20% and 40% [4,5].

Clinical manifestations of DKA include polyuria, polydipsia, weight loss, vomiting, abdominal pain, dehydration, Kussmaul breathing and altered sensorium. The severity of DKA is categorised based on arterial pH and serum bicarbonate levels into mild, moderate and severe, which also guide management strategies [6]. Timely diagnosis and standardised treatment protocols have led to a substantial reduction in DKA-related mortality, currently standing at less than 1% in developed countries [7]. However, complications such as cerebral oedema, shock, Acute Kidney Injury (AKI) and electrolyte imbalances continue to pose challenges, especially in critically ill children requiring intensive care [8]. Although rare, cerebral oedema is the most feared complication and remains the leading cause of DKA-related death in children [9].

The PICU plays a pivotal role in the management of moderate to severe DKA, where close monitoring of vital signs, fluid therapy, insulin infusion and correction of electrolyte disturbances are carried out meticulously [10]. Despite advances in therapy, the clinical profile and outcomes of children with DKA vary widely based on region, healthcare access and SES. A better understanding of clinical presentation, laboratory derangements and response to treatment in a tertiary care setting is essential to identify gaps in early recognition and to improve patient outcomes. Previous studies have examined the clinical profile and outcomes of DKA in different regions (North India and Dakshin Kannada) for patients up to 18 years of age [11,12].

The aim of this study was to retrospectively evaluate the clinical profile and outcomes of children with DKA admitted to the PICU in our tertiary care hospital in the northern part of West Bengal, Eastern India, thereby contributing to the regional data pool and guiding future clinical protocols.

Objectives:

1. To assess the clinical profile of children diagnosed with DKA, including presenting symptoms, biochemical parameters, precipitating factors and severity at admission.
2. To evaluate the outcomes of these patients in terms of recovery, complications, length of PICU stay, mortality and factors associated with mortality.

MATERIALS AND METHODS

This was a retrospective cohort study conducted at the PICU of Malda Medical College and Hospital, Malda, West Bengal, India,

after obtaining approval from the Institutional Ethics Committee (IEC) of Malda Medical College and Hospital on 08/05/2025 (certificate reference number: MLC-MC/IEC25/03). The study considered medical records from January 1, 2022, to December 31, 2024. The planning and execution of the study took place between January 2025 and March 2025, which included data retrieval, analysis and interpretation.

Inclusion criteria: Children under 12 years of age admitted to the PICU with a diagnosis of DKA between January 1, 2022 and December 31, 2024, were included in this study.

Exclusion criteria: Parents unwilling to enroll in the study, cases of transient hyperglycaemia and children diagnosed with inborn errors of metabolism were excluded from the study.

Sample size: The sample size was determined by the total number of eligible cases available during the defined study period, as this was a retrospective study. Using the census method, all children admitted to the PICU with a diagnosis of DKA who met the inclusion criteria from January 1, 2022, to December 31, 2024, were included. This resulted in a total of 40 cases. Including all available cases minimised selection bias and ensured that the study captured the complete clinical profile of DKA in the study setting during the specified timeframe.

Study Procedure

This study was conducted through a retrospective review of medical records of all eligible children admitted to the PICU with DKA during the study period. Information was extracted from hospital records, including admission sheets, clinical progress notes, laboratory reports and discharge summaries. Collected data included demographic information such as age, sex, SES [13], family history of diabetes mellitus and duration of symptoms. Clinical features and physical examination findings at admission were noted. Laboratory data, including blood glucose, HbA1c, serum electrolytes, blood urea, creatinine and arterial blood gas values, were reviewed. All patients were managed according to standard institutional and national guidelines for DKA [14]. Their treatment involved appropriate fluid management, insulin therapy, electrolyte correction and supportive care. Since the study involved secondary data from existing hospital records without direct contact with patients or families, a waiver of informed consent was obtained.

Operational definitions: DKA was defined as the presence of blood glucose levels greater than 200 mg/dL, arterial pH less than 7.3, serum bicarbonate levels below 15 mmol/L and the presence of ketone bodies in urine. Based on severity, DKA was further classified into three categories: mild DKA was characterised by a pH between 7.2 and 7.3, bicarbonate levels ranging from 16 to 20 mmol/L and patients being clinically alert but fatigued; moderate DKA was defined by a pH between 7.1 and 7.2, bicarbonate levels between 10 and 15 mmol/L and clinical signs such as Kussmaul respiration with drowsiness; severe DKA was diagnosed when the pH was below 7.1, bicarbonate was less than 10 mmol/L and the patient exhibited altered sensorium up to coma [15].

The AKI cases were defined in accordance with the Kidney Disease: Improving Global Outcomes (KDIGO) clinical practice guidelines. AKI was identified by any one of the following criteria: an increase in serum creatinine by more than 0.3 mg/dL within 48 hours, a rise in serum creatinine to more than 1.5 times the baseline value within seven days, or a reduction in urine output to less than 0.5 mL/kg/hour for six hours [16]. Cerebral oedema was diagnosed clinically based on symptoms such as altered consciousness, papilledema, hypertension, bradycardia, or supportive radiological findings [17].

STATISTICAL ANALYSIS

For statistical analysis, data were entered into Microsoft Excel and analysed using SPSS software (version 26). Descriptive statistics were calculated, including the mean and Standard Deviation (SD) for continuous variables and frequencies with percentages for categorical variables. Inferential statistical tests such as the Chi-square test or Fisher's exact test were employed for comparison of categorical data. Continuous variables were analysed using independent t-tests or the Mann-Whitney U test, depending on data distribution. A p-value <0.05 was considered statistically significant.

RESULTS

The highest percentage of cases belonged to the 9-12 years age group, with 17 (42.5%), followed by the 5-8 years age group, 13 (32.5%) and the 1-4 years age group, 10 (25%). The sex distribution was equal, with 20 males (50.0%) and 20 females (50.0%). The majority of children, 21 (52.5%), were from the low socio-economic group, followed by the middle group 15 (37.5%) and the high group 4 (10.0%). A positive family history of diabetes mellitus was observed in 11 children (27.5%), while 29 children (72.5%) had no known family history. Nearly half of the children, 18 (45.0%), presented with symptoms lasting 7-10 days, followed by 4-6 days, 13 (32.5%) and 1-3 days, 9 (22.5%). Most patients presented with severe diabetic ketoacidosis (DKA), 21 (52.5%), at the time of admission; fewer patients presented with mild DKA 9 (22.5%) and moderate DKA 10 (25%) [Table/Fig-1].

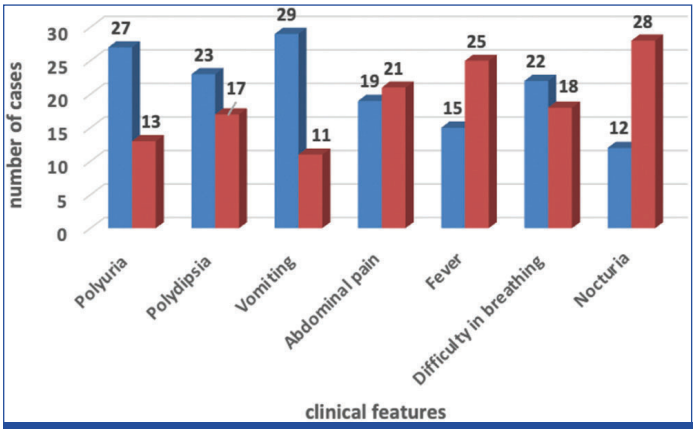
Parameters	n (%)
Age (years)	
1-4	10 (25.0)
5-8	13 (32.5)
9-12	17 (42.5)
Sex	
Male	20 (50.0)
Female	20 (50.0)
Socio-economic Status (SES)	
Upper	4 (10.0)
Upper middle	0
Middle	15 (37.5)
Lower middle	0
Lower	21 (52.5)
Family History	
Yes	11 (27.5)
No	29 (72.5)
Duration (Days)	
1-3	9 (22.5)
4-6	13 (32.5)
7-10	18 (45.0)
Severity of presentation	
Mild	9 (22.5)
Moderate	10 (25)
Severe	21 (52.5)

[Table/Fig-1]: Demographic and severity distribution of children with Diabetic Ketoacidosis (DKA) (n=40).
n: number of cases; DM: Diabetes mellitus

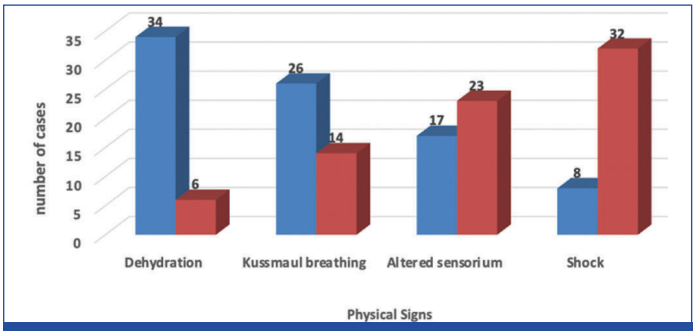
Clinically, polyuria was noted in 27 patients (67.5%), polydipsia in 23 (57.5%), vomiting in 29 (72.5%), abdominal pain in 19 (47.5%), fever in 15 (37.5%), difficulty in breathing in 22 (55%) and nocturia in 12 (30%) [Table/Fig-2].

Among the physical signs, dehydration was the most common finding, present in 34 patients (85.0%), followed by Kussmaul respiration in 26 (65.0%), altered sensorium in 17 (42.5%) and shock in 8 (20.0%) [Table/Fig-3]. Laboratory parameters are presented as mean±SD. All measurements were obtained at the time of admission, prior to the initiation of therapeutic interventions [Table/Fig-4]. The majority of patients, 16 (40.0%), stayed in the PICU for 1-3 days, with fewer staying 4-6 days, 13 (32.5%), or 7-10

days, 11 (27.5%) [Table/Fig-5]. With respect to outcomes, out of the total cases, 36 (90%) were successfully discharged, while 4 (10%) succumbed during treatment.



[Table/Fig-2]: Clinical features of children with Diabetic Ketoacidosis (DKA) at admission.
blue-present; red-absent



[Table/Fig-3]: Physical signs in children with Diabetic Ketoacidosis (DKA) at admission.
blue-present; red-absent

Parameter	Mean±SD	Normal range
Arterial blood Gas pH	7.12±0.14	7.35 to 7.45
Random blood sugar (mg/dL)	375.82±104.3	70-140
HbA1c (%)	5.61±0.70	<5.7
Serum urea (mg/dL)	42.62±25.42	7-20
Serum creatinine (mg/dL)	1.20±0.49	0.7-1.3
Serum sodium (mEq/L)	135.3±4.6	135-145
Serum potassium (mEq/L)	4.3±0.7	3.5-5

[Table/Fig-4]: Mean laboratory parameters at the time of admission.

PICU Stay (days)	n (%)
1-3	16 (40.0)
4-6	13 (32.5)
7-10	11 (27.5)

[Table/Fig-5]: Duration of PICU stay after admission.
PICU: Paediatric intensive care Unit; n: number of cases

A statistically significant association was noted with SES; 4 (100%) of expired cases belonged to the low SES group (p-value=0.004). Similarly, a positive family history of diabetes showed a significant relationship with outcome, as 3 (75%) of the expired group had a positive family history compared to only 8 (22.2%) among discharged patients (p-value=0.049) [Table/Fig-6]. Among the continuous variables, there was no significant difference in the duration of symptoms or PICU stay between the two groups. However, expired patients had significantly lower mean arterial pH (6.91±0.07 vs. 7.11±0.11; p-value=0.0016), higher HbA1c (6.98±1.01 vs. 5.51±0.61; p-value=0.0035), significantly elevated serum urea (78.50±37.91 vs. 39.93±18.97 mg/dL; p-value=0.0049) and serum creatinine (2.05±0.46 vs. 1.13±0.37 mg/dL; p-value=0.0021) compared to discharged patients. The true mean sodium lies

Parameter	Discharged n=36 (%)	Expired n=4 (%)	95% CI	p-value
Age group- 1-4	8 (22.2)	2 (50.0)	27.80% (-36.93 - 92.53)	0.271
Age group- 5-8	12 (33.3)	1 (25.0)	8.30% (-50.73 - 67.33)	0.410
Age group- 9-12	16 (44.4)	1 (25.0)	19.40% (-39.92 - 78.72)	0.416
Sex-boys	17 (47.2)	3 (75.0)	27.80% (-31.55 - 87.15)	0.298
Sex-girls	19 (52.8)	1 (25.0)	27.80% (-31.55 - 87.15)	0.298
SES revised-low	17 (47.2)	4 (100.0)	53.00% (22.81 - 83.19) Z	0.004*
SES revised-middle	15 (41.7)	0	41.70% (11.70 - 71.70) Z	0.137
SES revised-high	4 (11.1)	0	11.10% (-13.05 - 35.25)	0.429
Family history-no	28 (77.8)	1 (25.0)	52.80% (-5.64 - 111.24)	0.049*
Family history-yes	8 (22.2)	3 (75.0)	52.80% (-5.64 - 111.24)	0.049*

[Table/Fig-6]: Association between demographic characteristics and clinical outcome in children with DKA.
SES: Socio-economic status; CI: Confidence interval; p-value (<0.05): Significant, DKA: Diabetic ketoacidosis; Z-test for proportions applied for SES

between (133.7-154.0); the discharged group had higher sodium and the difference is significant (p-value=0.0001). Expired patients had significantly higher random blood sugar (RBS) levels (482.0±88.4 mg/dL vs. 358.11±82.84 mg/dL, p=0.04), while serum potassium was lower (3.46±0.46 vs. 3.86±0.45 mEq/L, p-value=0.07), though not statistically significant. These findings underscore the importance of metabolic derangements in predicting the outcome of paediatric DKA cases [Table/Fig-7].

Parameter	Discharged (n=36)	Expired (n=4)	95% CI	p-value
Duration of symptoms (days)	2.83±1.21	2.75±0.50	2.46-2.97	0.8193
ABG pH	7.11±0.11	6.91±0.07	6.95-7.09	0.0016
HbA1c (%)	5.51±0.61	6.98±1.01	5.60-6.65	0.0035
Serum urea (mg/dL)	39.93±18.97	78.50±37.91	45.60-72.36	0.0049
Serum creatinine (mg/dL)	1.13±0.37	2.05±0.46	1.19-1.91	0.0021
Serum sodium (mEq/L)	145.2±8.7	134.7±4.6	133.7-154.0	0.0001
PICU stay (days)	3.14±0.99	3.50±0.58	2.92-3.61	0.3170
RBS (mg/dL)	358.11±82.84	482.0±88.4	342.46-409.18	0.04*
Serum potassium (mEq/L)	3.86±0.45	3.46±0.46	4.076-4.524	0.07

[Table/Fig-7]: Comparison of continuous clinical and laboratory variables by outcome in children with DKA.
ABG: Arterial blood gas; PICU: Paediatric intensive care unit; Hba1c: Glycated haemoglobin; CI: Confidence interval; p-value (<0.05): Significant, DKA: Diabetic ketoacidosis; RBS: Random blood sugar

All four deaths (100%) were seen in severe DKA; no deaths occurred in mild or moderate cases (p-value=0.08). Analysis of symptoms revealed that abdominal pain (p-value=0.03), fever (p-value=0.01) and nocturia (p-value=0.008) were significantly associated with mortality, while other symptoms such as polyuria, polydipsia, vomiting and breathing difficulty did not show statistical significance. Regarding physical signs, altered sensorium (p-value=0.02) and shock (p-value=0.008) were strongly associated with poor outcomes, whereas dehydration and Kussmaul breathing did not demonstrate a significant association [Table/Fig-8].

Severity of disease	Discharged (36)	Expired (4)	p-value
Mild	9 (25%)	0	0.08
Moderate	10 (27.8%)	0	
Severe	17 (47.2%)	4 (100%)	
Symptoms			
Polyuria	23 (63.8%)	4 (100%)	0.28
Polydipsia	19 (52.7%)	4 (100%)	0.12
Vomiting	25 (69.4%)	4 (100%)	0.56
Abdominal pain	15 (41.6%)	4 (100%)	0.03*

Fever	11 (30.5%)	4 (100%)	0.01*
Breathing difficulty	18 (50%)	4 (100%)	0.60
Nocturia	8 (22.2%)	4 (100%)	0.008*
Physical signs			
Dehydration	31 (86.1%)	3 (16.7%)	0.49
Kussmaul breathing	23 (63.8%)	3 (17.4%)	1.00
Altered sensorium	13 (36.1%)	4 (100%)	0.02*
Shock	4 (11.1%)	4 (100%)	0.008*

[Table/Fig-8]: Association of disease severity, symptoms and physical signs with outcome.
*Significant

DISCUSSION

The present study evaluated 40 paediatric patients diagnosed with DKA, with the highest prevalence observed in the 9-12 years age group (42.5%), followed by 5-8 years (32.5%) and 1-4 years (25%). This age pattern partially aligns with findings from Atkilt HS et al., who reported a higher risk of DKA presentation in older children, especially those aged ≥9.5 years [18]. Similarly, Razavi Z and Hamidi F, found that the majority of their DKA patients were in the 10-14 years age group [19].

In the present study, the sex distribution was equal (M:F=1:1), which was comparable to Basavanthappa SP et al., who also observed a nearly equal gender distribution [20]. However, Razavi Z and Hamidi F, reported a higher risk for severe DKA among female patients, which was not reflected in present study cohort [19]. The SES showed a strong association with outcomes (p-value=0.004). All children from the low SES group expired, corresponding to an absolute risk difference of 53.0% (95% CI: 22.81% to 83.19%) compared to those discharged. The narrow lower bound above zero reinforces the reliability of this association and suggests that low SES is not just a chance finding but a clinically significant predictor of poor outcomes. No deaths occurred in the middle or high SES categories, where the confidence intervals (CIs) crossed zero, indicating no statistically significant difference compared to survivors. This pattern underscores the role of socio-economic disadvantage in delayed presentation, limited access to care and higher disease severity at admission. This was consistent with Basavanthappa SP et al., who reported that 88% of DKA cases were from lower socio-economic backgrounds, suggesting that delayed diagnosis and limited access to care in these populations contribute to worse outcomes [20].

A family history of diabetes was also significantly linked with outcomes (p-value=0.049). Children with a positive family history had a 52.8% higher mortality risk (95% CI: -5.64% to 111.24%) compared to those without such a history. Although the CI crosses zero, suggesting statistical uncertainty, the large effect size points toward a potentially meaningful clinical association, possibly due to genetic predisposition or a more aggressive metabolic profile. A similar trend was observed by Panakkal SJ et al., who noted a positive family history in 73.7% of their patients, although they did not report on its correlation with mortality [12]. PonJeba JMA and Varadarajan P, observed that major risk factors for presentation as DKA included a lack of family history of diabetes, shorter duration of illness and delayed diagnosis of DKA [21].

Clinically, vomiting (72.5%), polyuria (67.5%) and polydipsia (57.5%) were the most frequent presenting symptoms, mirroring previous reports. Razavi Z and Hamidi F, along with Basavanthappa SP et al., also reported polyuria and polydipsia in over 80% of cases, establishing these symptoms as common early indicators of DKA [19,20]. Physical findings, such as dehydration (85%) and Kussmaul respiration (65%), in the present study were also consistent with Razavi Z and Hamidi F, who noted altered sensorium in over 40% of patients—a comparable finding to the 42.5% with altered sensorium in our data [19].

The mean HbA1c in discharged patients was 5.51±0.61%, significantly lower than in those who expired (6.98±1.01; p-value =0.0035). This highlights poorer glycaemic control in the non survivors. Panakkal SJ et al., reported a much higher baseline mean HbA1c of 15.14±2.74%, possibly reflecting delayed diagnosis or chronic poor control in their cohort [12]. Despite the absolute difference in HbA1c values, both studies suggest that elevated HbA1c is a marker of worse outcomes.

Elevated serum urea and creatinine levels were significantly associated with mortality in our study (p-value=0.0049 and 0.0021, respectively), suggesting early renal involvement in severe DKA. Serum creatinine levels were also higher in patients who expired, with a 95% CI (2.05±0.46 mg/dL) closely aligning with their mean, indicating that renal impairment may be a contributor to adverse outcomes. These findings corroborate those of Basavanthappa SP et al., who identified renal failure as a key determinant of poor prognosis [20]. Additionally, Panakkal SJ et al., documented AKI in 10.5% of their patients, reinforcing the prognostic importance of renal function in DKA [12]. Alyahyawi N et al., concluded that there is a significant association between renal impairment and the severity of DKA [22]. Serum sodium levels were significantly higher in discharged patients compared to those who expired, with the 95% CI (133.7-154.0 mEq/L) not overlapping with the mean sodium of the expired group. This reinforces the association of lower sodium with poorer outcomes in paediatric DKA. A similar study conducted by PonJeba JMA and Varadarajan P, observed that higher sodium levels at admission are significant risk factors for mortality among children with diabetic ketoacidosis during initial presentation [21].

The mean duration of PICU stay was 3.14±0.99 days for discharged patients and 3.5±0.58 days for expired cases (p-value=0.317). The 95% CI (2.92-3.61 days) showed substantial overlap between the two groups, indicating that the length of the intensive care stay was not a distinguishing factor in outcomes. This was comparable to the average ICU stay of 3.5 days reported by Basavanthappa SP et al., [20]. However, Panakkal SJ et al., noted a longer average hospital stay of 7.42±3.27 days, possibly reflecting differences in severity or institutional protocols [12].

Elevated random blood sugar was significantly associated with mortality, indicating that hyperglycaemia is an adverse prognostic factor. Van Waardenburg DA et al., reported that hyperglycaemia in critically ill children was independently associated with increased mortality and adverse outcomes, consistent with present study findings [23]. While lower potassium levels were observed in expired cases without statistical significance, the trend underscores the importance of vigilant electrolyte monitoring in critically ill children. Kitabchi AE et al., highlighted that hypokalaemia is a frequent complication of DKA, resulting from osmotic diuresis and insulin therapy and may contribute to poor outcomes if not promptly corrected [24]. The overall mortality rate in this study was 10%, which was lower than the 11.5% reported by Basavanthappa SP et al., [20]. In contrast, Panakkal SJ et al., reported no mortality, suggesting better outcomes with prompt and effective management, although their sample size was smaller [12]. In present study, lower mortality rate may also reflect timely recognition and early PICU intervention in a tertiary care setting.

This study highlights that certain clinical features at presentation can predict adverse outcomes in paediatric patients. Key indicators, including abdominal pain, fever, nocturia, altered sensorium and shock, were significantly associated with mortality, emphasising their role as potential warning signs. Jayashree M and Singhi S, similarly identified shock and altered mental status as strong predictors of poor outcomes, underscoring the need for early recognition and aggressive management in such cases [25]. Conversely, common metabolic symptoms such as polyuria, polydipsia and vomiting did not correlate with mortality, suggesting that systemic complications rather than initial metabolic symptoms drive adverse outcomes.

Limitation(s)

This study had certain limitations that should be acknowledged. The relatively small sample size (n=40) limits the statistical power and may reduce the ability to detect subtle associations between demographic or clinical factors and outcomes. As a single-centre study conducted in a tertiary care hospital, the findings may not be generalisable to community or primary care settings where presentation patterns and access to care may differ. The retrospective design relies on the accuracy and completeness of medical records, which may introduce information bias, particularly in the recording of symptom onset, socio-economic status and family history.

CONCLUSION(S)

The present study underscores that DKA in children is more frequently observed in the 9-12 years age group, with a notable proportion arising from lower socio-economic backgrounds. Key clinical manifestations, such as dehydration, vomiting and altered sensorium, were prominent among affected patients. The study found that mortality was significantly associated with severe metabolic derangements, including low arterial pH, elevated HbA1c and raised serum urea and creatinine levels. Additionally, low SES and positive family history of diabetes were notable risk factors for poor outcomes. These findings reinforce the need for early recognition, prompt intervention and improved community awareness to reduce morbidity and mortality associated with paediatric DKA. Present study contributes to the existing literature by providing demographic, clinical, laboratory and precipitating factors from this geographical area. However, the small sample size limits the generalisability of present study findings. To better determine the contributing factors, including disease severity, there is a need for prospective studies with larger sample sizes from this area.

REFERENCES

[1] Wolfsdorf JI, Glaser N, Agus M, Fritsch M, Hanas R, Rewers A, et al., ISPAD Clinical Practice Consensus Guidelines 2018: Diabetic ketoacidosis and the hyperglycemic hyperosmolar state. Pediatr Diabetes. 2018;19(Suppl 27):155-77. Doi: 10.1111/pedi.12701. PMID: 29900641.

[2] Dabelea D, Mayer-Davis EJ, Saydah S, Imperatore G, Linder B, Divers J, et al. Prevalence of type 1 and type 2 diabetes among children and adolescents from 2001 to 2009: SEARCH for Diabetes in Youth Study. JAMA. 2014;311(17):1778-86. Doi: 10.1001/jama.2014.3201. PMID: 24794371; PMCID: PMC4368900.

[3] Usher-Smith JA, Thompson MJ, Sharp SJ, Walter FM. Factors associated with the presence of diabetic ketoacidosis at diagnosis of diabetes in children and young adults: A systematic review. BMJ. 2011;343:d4092. Doi: 10.1136/bmj.d4092. PMID: 21737470; PMCID: PMC3131115.

[4] Dayal D, Saini AG, Attri B. Clinical profile and outcome in children with diabetic ketoacidosis: A study from a tertiary care center in North India. Indian J Endocrinol Metab. 2014;18(5):632-4.

[5] Kalra S, Unnikrishnan AG, Baruah MP. Management of diabetic ketoacidosis in children: An Indian perspective. Indian J Endocrinol Metab. 2012;16(Suppl 2):S144-51.

[6] Glaser N, Barnett P, McCaslin I, Nelson D, Trainor J, Louie J, et al. Risk factors for cerebral edema in children with diabetic ketoacidosis. N Engl J Med. 2001;344(4):264-9. Doi: 10.1056/NEJM200101253440404. PMID: 11172153.

[7] Craig ME, Hattersley A, Donaghue KC. Definition, epidemiology and classification of diabetes in children and adolescents. Pediatr Diabetes. 2009;10(Suppl 12):3-12. Doi: 10.1111/j.1399-5448.2009.00568.x. PMID: 19754613.

[8] Rewers A, Klingensmith G, Davis C, Petitti DB, Pihoker C, Rodriguez B, et al. Presence of diabetic ketoacidosis at diagnosis of diabetes mellitus in youth: The SEARCH for Diabetes in Youth Study. Pediatrics. 2008;121(5):e1258-66. Doi: 10.1542/peds.2007-1105. PMID: 18450868.

[9] Muir AB, Quisling RG, Yang MC, Rosenbloom AL. Cerebral edema in childhood diabetic ketoacidosis: Natural history, radiographic findings and early identification. Diabetes Care. 2004;27(7):1541-6. Doi: 10.2337/diacare.27.7.1541. PMID: 15220225.

[10] Al-Sayed NA, Aleem A, Al-Daghri N, Al-Orifi F, Al-Muhsen S, Al-Shehri A, et al. Management and outcomes of diabetic ketoacidosis in children and adolescents at a tertiary care center in Saudi Arabia. Diabetes Res Clin Pract. 2010;90(3):e45-6. Doi: 10.1016/j.diabres.2010.08.006.

[11] Bhardwaj P, Yadav V, Sharma M. Clinical profile and outcome of the children with diabetic ketoacidosis in a hilly Himalayan state of North India. Int J Res Med Sci. 2017;5:5402-05.

[12] Panakkal SJ, Shirodkar D, Saldanha PRM. Clinical profile and outcome of children presenting with diabetic ketoacidosis at a tertiary care hospital in Dakshina Kannada. Int J Contemp Pediatr. 2023;10:644-48.

[13] Anand A, Mandal I, Hossain S. B.G. Prasad Scale 2025: An updated framework for socioeconomic assessment in India. Natl J Community Med. 2025;16(5):555-58. Doi: 10.55489/njcm.160520255339.

[14] British Society for Paediatric Endocrinology and Diabetes. BSPED guideline for the management of children and young people under the age of 18 years with diabetic ketoacidosis—2021. London: BSPED; 2024. Available from: https://www.bsped.org.uk/clinical-resources/bsped-dka-guidelines/.

[15] Kliegman RM, St Geme JW III, Blum NJ, Tasker RC, Wilson KM, Schuh AM, et al., editors. Diabetes mellitus. In: Nelson Textbook of Pediatrics. 22nd ed. Vol. 2. Philadelphia: Elsevier; 2024. p. 3524.

[16] Khwaja A. KDIGO clinical practice guidelines for acute kidney injury. Nephron Clin Pract. 2012;120(4):c179-84. Doi: 10.1159/000339789. PMID: 22890468.

[17] Kliegman RM, St Geme JW III, Blum NJ, Tasker RC, Wilson KM, Schuh AM, et al., editors. Diabetes mellitus. In: Nelson Textbook of Pediatrics. 22nd ed. Vol. 2. Philadelphia: Elsevier; 2024. p. 3527.

[18] Atkilt HS, Turago MG, Tegegne BS. Clinical characteristics of diabetic ketoacidosis in children with newly diagnosed type 1 diabetes in Addis Ababa, Ethiopia: A cross-sectional study. PLoS One. 2017;12(1):e0169666. Doi: 10.1371/journal.pone.0169666. PMID: 28135278; PMCID: PMC5279745.

[19] Razavi Z, Hamidi F. Diabetic ketoacidosis: Demographic data, clinical profile and outcome in a tertiary care hospital. Iran J Pediatr. 2017;27(3):e7649. Doi: 10.5812/ijp.7649.

[20] Basavanthappa SP, Pejaver R, Raghavendra K, Srinivasa V, Suresh Babu MT. Clinical profile and outcome of diabetic ketoacidosis in a tertiary care hospital in South India. Int J Contemp Pediatr. 2015;2(1):29-31. Doi: 10.5455/2349-3291.ijcp20150207.

[21] PonJeba JMA, Varadarajan P. DKA as initial presentation in children with type 1 diabetes mellitus-risk factors: A case control study. Int J Contemp Pediatr. 2018;5(3):815-18. Doi: 10.18203/2349-3291.ijcp20181258.

[22] Alyahyawi N, Alghamdi D, Almahmoudi A, Sandokji I, Bokhari S, Safdar OY, et al. Risk factors of acute kidney injury in children with diabetic ketoacidosis. BMC Nephrol. 2025;26:254.

[23] Van Waardenburg DA, Jansen TC, Vos GD, Buurman WA. Hyperglycemia in children with meningococcal sepsis and septic shock: The relation between plasma levels of insulin and inflammatory mediators. Crit Care Med. 2006;34(6):1633-38. Doi: 10.1097/01.CCM.0000215836.21273.2D.

[24] Kitabchi AE, Umpierrez GE, Miles JM, Fisher JN. Hyperglycemic crises in adult patients with diabetes. Diabetes Care. 2009;32(7):1335-43. Doi: 10.2337/dc09-9032.

[25] Jayashree M, Singhi S. Diabetic ketoacidosis: Predictors of outcome in a pediatric intensive care unit of a developing country. Pediatr Crit Care Med. 2004;5(5):427-33. Doi: 10.1097/01.PCC.0000137986.16021.79.

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